Foreign Animal Disease Report

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Emergency Programs

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In This Issue--

Current Events

Puerto Rico Tick Program

The tick eradication program for Boophilus microplus and Amblyomma variegatum in Puerto Rico has been making substantial gains. Total premises freed of ticks exceeded 6,000 by the second quarter of fiscal year (FY) 1985 (March 31, 1985) with more than 6,000 herds to be added for a total of more than 12,000 free herds by the end of the FY (September 30, 1985).

Tick problems in Puerto Rico were complicated by the diagnosis of babesiosis (cattle tick fever) in April 1985.

A veterinarian was called on April 1, 1985, to treat animals at one of four dairy herds with 1,200 cattle. Specimens were submitted to National Veterinary Services Laboratories (NVSL). Blood smears from this material revealed Babesia bovis-like bodies. NVSL sent two veterinarians to Puerto Rico to investigate. They prepared smears which revealed B. bovis and confirmed the diagnosis of babesiosis on April 29, 1985.

The signs seen in this herd were sudden death, temperature in excess of 105°F, ruminal stasis, accelerated heart and respiratory rates, depression, wine-colored urine, and anorexia. Post-mortem examination revealed rose-colored fluid and large blood clots in the abdominal cavity, and rose-colored fluid in other body cavities. The peripheral blood was watery and would not clot. The spleen was engorged.

By August 8, 1985, 262 premises were under quarantine because of babesiosis or exposure to vector ticks. Fourteen herds were found positive upon examination of blood smears and 108 herds were seropositive. (Dr. Glen O. Schubert, 436-8438).

VVND Update

A young yellow-naped Amazon parrot was found to be infected with velogenic viscerotropic Newcastle disease (VVND) in North Carolina. This bird was recently purchased from a wholesale

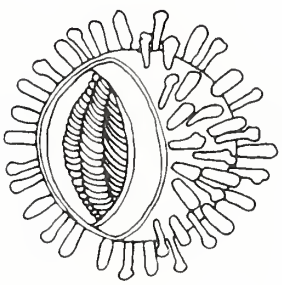
distributor in Cape Girardeau, Missouri. Specimens collected at the Missouri premises contained VVND virus.

Velogenic viscerotropic Newcastle disease viruses were also isolated from a dead yellow-naped Amazon parrot in Phoenix, Arizona, and a live one in California. The owners of these birds could not be located. (Dr. K. A. Hand, 301 436-8065)

Vesicular Stomatitis in Arizona New Mexico, and Colorado

New Jersey type vesicular stomatitis was diagnosed in Arizona and New Mexico during June and Colorado in July 1985 (see 13-1). The virus was isolated from both cattle and horses in New Mexico and Colorado. Over 50 premises there were confirmed as affected by the disease. Horses on two premises were affected in Arizona. Epidemiological investigations are in progress, including studies of possible sources of the virus responsible for this outbreak. (Dr. K. A. Hand, 301 436-8065)

Avian Influenza Update



Now that the H5N2 influenza virus (AI) has been eradicated from domestic poultry in the United States (see 13-2), we can ask what we have learned and whether such an event can be prevented in the future. The H5N2 virus was first isolated from Pennsylvania chickens in April 1983, when it caused mild respiratory infection and low mortality. We do not know how long this virus had been circulating in domestic poultry in Pennsylvania or elsewhere. If it was present earlier, it caused inapparent infections at a very low incidence. Subsequently, in October 1983, the virus became highly virulent and was directly or indirectly responsible for the destruction of over 17 million birds at a cost of over $60 million for the eradication program alone.

Several questions remain unresolved. These include: What is the source of the virus, how did it become highly virulent, and what is the likelihood of another outbreak of virulent AI?

Analysis of the nucleic acids of the H5N2 influenza virus by ribonucleic acid (RNA) hybridization indicate that one or more of the eight RNA segments were most closely related to the RNA segments of influenza viruses isolated from wild ducks, domestic turkeys, or gulls. At this time, no single influenza virus has been identified as the progenitor of the H5N2 strain. The analysis leaves little doubt that the virus originated from an avian species, but whether it was from a domestic turkey, wild duck, or some other aquatic bird remains unknown. It seems unlikely that we will be able to establish the source of the avirulent H5N2 influenza virus that initiated the epidemic in Pennsylvania.

Surveillance of wild birds in Pennsylvania during 1984 disclosed a high frequency of influenza viruses in wild ducks. The peak of virus activity in wild ducks was in August 1984, when one H5N2 virus was isolated. Although this virus was antigenically similar to the virus causing the epidemic in Pennsylvania, detailed genetic and sequence analysis enabled us to say with certainty that this virus was not a member of the Chicken/Pennsylvania H5N2 "family."

Two possibilities were considered to explain how the H5N2 virus became highly pathogenic. One possibility was genetic reassortment between the initial avirulent H5N2 strain and another influenza virus. Another possibility was acquisition of virulence by the accumulation of point mutations. Nucleic acid analysis by oligonucleotide mapping showed only a small number of differences between the avirulent and virulent strains, indicating that reassortment or genetic recombination was not involved in the acquisition of virulence. The small number of differences indicated that point mutations were probably important.

Previous studies have indicated that the hemagglutinin (HA) is important in the virulence of the H5 and H7 influenza viruses. For a virus to be virulent, it must be able to produce plaques in chick embryo fibroblast tissue cultures without trypsin. Also, the hemagglutinin (HA) molecule must be cleaved into HA1 and HA2 in the tissue culture system. The H5N2 virus isolated in October 1983 fulfilled each of these requirements. Sequence analysis of the HA gene of the prototype avirulent H5N2 virus isolated in April 1983 and the virulent strains showed that there were four amino acid changes in the HA between the avirulent and virulent strains. In order to determine which of these changes was really important and to learn more about the evolution of the Chicken/Pennsylvania virus, the hemagglutinin gene of additional virulent and avirulent H5N2 viruses was sequenced.

These studies showed that the virulent Chicken/Pennsylvania/83 (H5N2) influenza viruses had two amino acid differences from the avirulent viruses. They also indicated that the virulent and avirulent virus originated from a common ancestor, and that there was a single introduction of virulent virus into chickens. Sequence analysis also showed that an H5N2 virus isolated in December 1984 from chickens in Washington, D.C., is a member of the Chicken/Pennsylvania/83 family of viruses and that the viruses in turkeys in Virginia were derived from the avirulent family of viruses.

The virulence of influenza viruses has been associated with two or more of the eight RNA segments of influenza viruses, one of which is the hemagglutinin gene. We can ask which of the other genes in H5N2 is important. In order to answer this question, reassortant viruses have been prepared in the laboratory. The hemagglutinin gene from the virulent H5N2 strain has been switched to the avirulent strain and vice versa. Although these studies are incomplete, initial observations indicate that seven gene segments from the avirulent virus plus the hemagglutinin gene from the virulent virus are sufficient to make the avirulent virus fully virulent. The implication of this finding is that the avirulent virus was a "disaster waiting to happen." All that appears to be required is a single point mutation at a critical residue in the hemagglutinin to convert the avirulent virus to a fully virulent virus. The April 1983 avirulent virus contained the other genes necessary for virulence.

During the eradication program, questions were raised concerning the eradication of the avirulent H5N2 influenza virus from poultry in Pennsylvania, and more particularly from turkeys in Virginia. The molecular studies emphasize that the avirulent virus had the potential to become a highly dangerous virus.

Defective interfering (DI) particles were found in the avirulent H5N2 viruses isolated before October 1983 from chickens in Pennsylvania. These are influenza virus particles that are defective and cannot replicate without parental virus. The presence of small molecular weight RNA's in addition to the eight standard RNA's was indicative of DI particles. In experimental studies, it was shown that the early avirulent viruses containing defective interfering particles could interfere with the virulence of the highly pathogenic strains. If a virus containing DI particles was mixed with the fully virulent virus, only a limited number of chickens were killed, whereas the virulent virus alone killed 100 percent of the inoculated chickens. The significance of these findings is still not clear and further work is required. Although considerable work has been done on laboratory strains of influenza virus and DI particles, the demonstration of DI particles in H5N2 virus from birds was one of the first illustrations that DI virus particles may occur in nature. We do not know if the DI particles played any role in preventing or promoting the emergence of virulent H5N2 influenza virus.

The likelihood of another outbreak of highly virulent influenza virus in domestic poultry is now as high as it was before the epidemic in Pennsylvania in 1983. There is a perpetual reservior in aquatic birds of all of the known influenza viruses. Most influenza viruses are nonpathogenic in their natural host and domestic poultry; however, a certain number of these avirulent viruses can replicate in domestic poultry, thus opening the door for mutation and adaptation.

Several recent incidents emphasize the potential of "virulent" influenza viruses. In 1983, viruses of the subtype H7N7 caused 30 percent mortality in harbor seals in Massachusetts; in 1983-84 H5N2 virus caused high mortality in chickens in Pennsylvania; in 1983, a virulent H5N8 appeared in turkeys in Ireland and an antigenically similar virus was isolated from ducks; and in 1985, a highly virulent H7N7 appeared in chickens in Australia. It is clear that reservoirs of virulent H5 and H7 influenza viruses exist in nature. These viruses are probably avirulent in their natural hosts, and become highly virulent in domestic avian species either by mutation, as occurred in the Chicken/Pennsylvania H5N2 incident, or by direct transfer between species, as appears to have occurred in the Turkey/Ireland H5N8 incident.

A practical step which can be taken to minimize the likelihood of another AI epidemic is to prevent direct or indirect contact between aquatic birds and domestic poultry. Aquatic birds, such as ducks, should be discouraged from inhabiting ponds near domestic poultry raising areas. Influenza viruses usually replicate in the cells lining the intestinal tract of the

natural host and are shed in high concentrations in feces. A contaminated water supply is the optimal method of transmission. Good sanitation and husbandry practices can completely prevent the introduction of influenza viruses into properly maintained poultry houses.

A highly pathogenic influenza virus may be defined as one that results in not less than 75 percent mortality in 8 days in at least eight healthy, susceptible chickens, 4-8 weeks old, inoculated by the intramuscular, intravenous, or caudal air sac route with bacteria-free infectious allantoic or cell culture fluids. This definition was established at the First International Symposium on Avian Influenza at Beltsville in 1983 and has served a useful purpose. During the H5N2 influenza epidemic, it was apparent that some strains of H5N2 virus were of intermediate virulence. The age and strain of birds used was important in determining whether or not the virus was pathogenic. In the future, it will be necessary to reevaluate the criteria for defining virulent virus. Criteria that should be considered are:

1. The ability of virulent influenza viruses to produce plaques in chick embryo fibroblast cultures in the absence of trypsin.

2. Cleavage of the hemagglutinin molecule into HA1 and HA2 in chick embryo fibroblast tissue cultures in the absence of trypsin.

3. High mortality in the domestic species of virus origin after natural routes of inoculation.

4. A mean death time of less than 2 days in chicken embryos incubated at $35^{o}C$.

A combination of these properties would be useful in defining a highly pathogenic influenza virus.

In summary, available evidence indicates that the H5N2 influenza virus in poultry in Pennsylvania originated from an avian source, and that the virus became virulent by a limited number of mutations in the hemagglutinin gene. The avirulent H5N2 strain was potentially a dangerous virus, because gene segments of the virus necessary for high virulence were already present.

The gene pool of influenza viruses in aquatic avian species remains high, particularly at certain times of the year, making it mandatory to minimize contact between domestic and wild avian species. The practice of good husbandry and hygiene can greatly reduce the possibility of another AI epidemic. (Robert G. Webster, Yoshihiro Kawaoka, and William J. Bean, St. Jude Children's Research Hospital, P.O. Box 318, Memphis, Tennessee 38101, 901 522-0415)

African Swine Fever in Belgium

African swine fever (ASF) appeared for the first time in West Flanders, Belgium, on March 8, 1985. This disease has caused extensive losses in Spain, Portugal, and Sardinia since the 1960's.

Belgian officials have established the source of their outbreak as an uncooked head cheese type pork product called "Tete du Porc" brought from Spain to Belgium by a man living in a house adjacent to the first affected herd (index case). He reportedly threw a portion of this product into a boar pen sometime between Christmas and the New Year. Inclement weather at that time apparently delayed consumption of the meat. The boar first showed lethargy, fever, and dehydration on January 30 or 31 and responded temporarily to antibiotic treatment before dying. The attending veterinarian did not suspect a contagious disease.

Hog Cholera (HC) was suspected in the area in late February but was ruled out by tests conducted at the veterinary school in Ghent. Specimens were then taken to the virus diagnostic laboratory in Alfort, France, where ASF virus was identified.

This first incursion of ASF in Belgium was accompanied by an absence of early clinical evidence of spread, low morbidity of 16 percent in the first herd affected, and similarity of the disease to the hog cholera already endemic in the area.

Spread of the disease to eight additional farms was attributed to visits by the attending veterinarian to the farms of four other clients and to the movement of feeder pigs to four other premises. A total of 5,400 swine were on the first nine farms affected.

Eighteen additional premises were identified which had purchased feeder pigs from affected premises. The decision was made to slaughter all 19,500 infected or exposed swine in order to prevent further spread of disease.

Disposal required the movement of sick and exposed swine in specially designed trucks to an approved rendering facility in the affected area. The Government of Belgium and the European Economic Community (EEC) equally shared the cost of indemnity payments to owners.

To stop the further spread of ASF, no swine movements within or from an area of 50,000 square kilometers were allowed for 2 weeks. About 50 slaughter plants located in this area were also closed down for the same period of time. By April 18, 1985, EEC agreed to reduce the control area to about one-third its original size.

Belgian disease control guidelines require that all pork from the original control area outside the newly designated small control area be consumed within Belgium or be fully cooked before being moved out of the country. Fresh pork has been placed in cold storage in the control area since the ASF campaign began. New guidelines for the processing of this meat,

which would be acceptable to all EEC member countries, are being developed.

Through a system of serological surveillance, ASF antibodies were found on May 15, 1985, in 10 pigs on a premises with 350 fattening swine. The owner had 15 different swine feeding premises, two of which were also affected by ASF. There was extensive human movement between farms. A total of 30 premises with about 14,000 swine have had direct or indirect contact with the ASF exposed animals. The boundary of the control area was extended northward by several kilometers to include all affected swine. (Adapted from a report received from Dr. Claude J. Nelson, Veterinary Services Representative, U.S. Embassy, Rome, Italy)

Foot-and-Mouth Disease in Italy

Foot-and-mouth disease (FMD) was diagnosed in Italy on November 27, 1984. The first confirmed case involved a flock of 600 sheep, nearly all with extensive lesions. Although no sheep died of FMD, the entire flock was immediately destroyed to stop the spread of virus. Within the next 10 days, 24 more cases were found in the same region, primarily in cattle.

Modena, Italy, the area where these cases were found, has one of the largest livestock markets in Europe for breeding cattle. This market was built in 1951 and serves brokers, dealers, or owners from several countries. As a result of the FMD outbreak, the market was closed for the first time since its opening, but was reopened on January 21, 1985. By the end of May 1985, FMD had been reported in 11 of the 19 regions of Italy and the island of Sicily, with 135 cases confirmed. The disease was spread from the Po Valley west to an area near the French border and south into central Italy. No cases have been reported along borders with Austria and Yugoslavia or on the island of Sardinia.

The Italian FMD program has for years called for twice a year compulsory vaccination using AOC trivalent vaccine made in Italy. All cattle over 3-4 months of age are required to be immunized. This program was effective until 1984 when the epizootic started. The numbers of animals vaccinated reportedly have declined in the last year or two.

Virus strain A_5 (Modena) was isolated and typed early in the outbreak. Since all vaccinated cattle were supposed to have good immunity to vaccine strain A_5 (Parma 1962), this led to early speculation that the virus involved in the outbreak may be a variant. Vaccine trials were conducted in Italy and typing was carried out at Pirbright, England, and in Italy. Results showed that vaccine produced in Brescia, Italy, provided complete protection against the virus being isolated from this outbreak even when only one-sixteenth of a standard bovine dose was given. Laboratory assays showed the A_5 strains were either very similar or identical.

The question of the origin of virus in this epizootic is still being debated. No connection has been established between the

Italian outbreak and the importation of meat or live animals.

Responsibility for establishing regulatory policy and control measures is borne by separate regional and Provincial governments. The general policy has been to destroy all cattle showing clinical lesions or fever. Most swine, sheep, and goats associated with an outbreak have also been destroyed. Cattle remaining on infected premises are then vaccinated. By the end of April, 2,096 cattle, 8,051 swine, and 681 sheep and goats either died of FMD or were destroyed in efforts to control the spread of disease.

In cattle, the morbidity rate has remained at 14 percent since the beginning of the outbreak. In swine, this rate was much higher during the first months, with several herds having rates of 80 percent or higher. The swine morbidity rate has dropped considerably to an average of 10 percent during recent months. It is interesting to note that of the 15 affected premises with sheep and goats, only one with 600 head had sheep with signs of FMD.

Mortality rates through April have been 0.1 percent (11 out of 11,860) in cattle, 0.2 percent (61 out of 27,796) in swine, and none (692 at risk) in sheep and goats. Highest losses were reported on swine in the Piemonte region, where 638 of a total of 798 animals showed signs and 60 died. In another instance in the same area, 1,196 of a total 1,495 swine had FMD signs but none died.

A new outbreak in a herd of 1,300 cattle in the Po Valley was confirmed on June 5, indicating that FMD virus continues to exist in northern Italy. (Adapted from a report received from Dr. Claude J. Nelson, Veterinary Services Representative, U.S. Embassy, Rome, Italy)

World Animal Disease Roundup

Just about the time authorities began to breathe a little easier about the African swine fever (ASF) situation in Belgium, the disease made a comeback and was found on three new premises, all in the area originally designated as quarantine Zone I. Two of these were detected by serological means, and the third by clinical diagnosis. Thirty-two herds with 14,000 pigs were destroyed to stop further spread. (See article in this issue on ASF in Belgium.)

Foot-and-Mouth disease is again being reported from all endemic areas in South America, with Argentina having the most cases. Chile has had no outbreaks for over a year and is again being considered for recognition as free from this disease. With the exception of Malawi, where one case of type O was reported, no cases have been reported from Africa. Of interest are reports on type $Asia_1$ from Israel, Malaysia, southern Thailand, and Bahrain. In Europe, the type A epidemic in Italy continues. (See article in this issue on FMD in Italy.)

Sheep and Goat Pox, unreported for some time, was again seen in Mali, Cyprus, Morocco, and Israel.

Reports on **rinderpest** were received from Mali and Ivory Coast in Africa, Bahrain on the Persian Gulf, and Iraq, where the disease is supposed to have killed several thousand cattle.

Of interest is an outbreak of avian **influenza** in Australia. The virus causing this problem was identified as type H7N7, the cause of classical fowl plague. Fortunately, the Australians were able to quickly control the situation. (Dr. Hans J. Seyffert, 301 436-8285).

Audiovisuals

Veterinary Services maintains motion pictures, slides, slides with audiotapes, and videotapes to inform veterinarians, livestock producers, and others on the nature of major foreign diseases of livestock and poultry. Disease characteristics that signal a new outbreak are emphasized, along with information on prevention and control. Locations from which VS audiovisuals may be borrowed or purchased have been changed to the following:

1. Color motion pictures, 16-millimeter, English (E) or Spanish (S) soundtrack, can be purchased from WRS Motion Picture and Video Laboratory, 210 Semple Street, Pittsburgh, Pennsylvania 15213 (telephone 412 687-3700). These films were produced during the 1970's. Purchase prices range from $44.42 to $73.90, payable to WRS laboratory:

	Minutes running time
Foot-and-mouth disease (E, S)	15
Exotic Newcastle disease (E)	11
Malignant catarrhal fever (E, S)	12
Ephemeral fever (E, S)	13
Sheep pox and goat pox (E, S)	11
African horsesickness (E)	11
African swine fever/hog cholera (E, S)	13
Contagious bovine pleuropneumonia (E, S)	9
Swine vesicular disease (E, S)	8

2. Color motion pictures, 16-millimeter, English sound track, for purchase or rental from General Services Administration, National Archives and Records Service, National Audiovisual Center, Washington, DC 20409 (telephone 301 763-1896).

	Minutes running time
Bovine contagious pleuropneumonia (1956)	28
Exotic Newcastle disease (1973)	22
Vesicular exanthema (1973)	17

A limited number of these films and a slide-tape set on "Preventing African swine fever" are available for loan from Veterinary Services, Animal Health Programs (telephone 301 436-8363).

Focus on... Jembrana Disease

Jembrana disease, an infectious noncontagious disease named for its appearance in the Jembrana district of Bali Island, Indonesia, was first reported in December 1964 as an epizootic of Bali cattle, which are domesticated Banteng cattle (Bos javanicus) and buffaloes. An estimated 60 percent of the total cattle and buffalo populations of Jembrana district were affected. The mortality rate was approximately 99 percent. The disease reappeared in 1965-66 and 1966-67 with mortality rates of approximately 95 and 67 percent, respectively. Total losses were estimated to be 60,000 head of cattle and buffaloes during the period December 1964-September 1967.

Early studies of the clinical and pathological features suggested that the disease might be rinderpest. In a mass vaccination campaign in 1966-67, an estimated 88 percent of the animals at risk were given lapinized rinderpest vaccine. No new epizootics of Jembrana disease were reported during the following 4 years.

Tabanan Disease

In April 1972, Jembrana reappeared in a less severe form, affecting only Bali cattle in the neighboring Tabanan district. Dr. Iwan T. Budiarso and Dr. S. Hardjosworo studied the new outbreak under the name of "Tabanan disease." They concluded that Jembrana disease is a new entity and not rinderpest. The authors believed that it could be a rickettsial disease because of the presence of hemorrhages and endothelial lesions, production of an orchitis during transmission trials in guinea pigs, and rickettsia-like inclusion bodies in infected cells.

In 1974 a new epizootic occurred in Bali. A United Nations Development Program-Food and Agriculture Organization (UNDP-FAO) team headed by Dr. S. Ramachandran and Dr. H. P. Harding began studying the disease. These investigators concluded that Jembrana disease is probably of viral origin and stated that the rickettsia hypothesis could not be confirmed by their investigations. They felt the hypothetical virus attacked lymphocytes, monocytes, platelets, and endothelial cells, and produced nuclear changes similiar to those of neoplasia, but without permanent neoplastic transformation.

Rama Dewa Disease in Sumatra

In May 1976 an outbreak of "Rama Dewa" occurred at Lampung, South Sumatra. Clinically and pathologically similar to Jembrana disease, Rama Dewa disease affected only Bali cattle. The Government of Indonesia (GOI) has been relocating farmers from the populated areas of Java and Bali to the island of Sumatra in a program in which many Bali cattle have been sent along with farmers from Bali to Sumatra. At present Bali cattle are allowed to be exported from Bali only for slaughter in Jakarta.

Suspected outbreaks of Jembrana disease occurred in November 1978 in the Banyuwangin district of East Java. Except for Sumatra and East Java all reports of Jembrana have been from Bali Island.

On Bali, the general incidence of the disease declined during 1976-80 but became epizootic again in 1981, with more than double the reported cases and nearly five times the number of deaths. The number of cases and deaths reported is as follows:

Year	Cases	Deaths
1974	4,584	336
1975	4,610	345
1976	1,970	203
1977	1,188	125
1978	1,739	55
1979	1,851	144
1980	1,736	121
1981	4,100	560
1982	881	number not available

Fewer cases of Jembrana disease were reported in 1983-84 and the disease appeared in a less severe form than seen earlier.

Clinical Signs

Clinical signs of Jembrana disease include anorexia, fever of 104°-107.6°F (40°-42°C) which lasts 3-6 days, generalized lymphadenopathy with enlargement of the prescapular and prefemoral lymph nodes up to 10 or 20 times normal size, nasal discharge, and increased salivation. Infected animals have diarrhea or dysentery. "Blood sweating"--free blood on the surface of the skin--has often been reported during the febrile period in acute cases. Mucosal erosions of the lips, gums, tongue, and pharynx are sometimes seen during the febrile period. Ocular and nasal discharge as well as conjunctivitis may be present. Hemorrhages are occasionally observed in the vaginal mucosa, base of the tongue and in the anterior chamber of the eye. Pregnant animals may abort. Leucopenia is a consistent hematological finding early in the disease as is anemia with formations of large monocytes often containing rickettsia-like organisms (RLO). These RLO are seen in monocytes and not in granulocytes as in Ondiri disease. Relapses have been reported in recovered cattle. In general, relapse fevers are of shorter duration and the peak temperatures are significantly lower than initial fevers. Leucopenia is also of shorter duration and of lower intensity during relapses than during the initial sickness. During the last few years Jembrana disease has presented a much milder clinical and pathological picture. The hemorrhagic diarrhea and "blood sweating" previously reported are rarely seen and the morbidity and mortality rates are greatly reduced.

Pathology

The most important changes associated with Jembrana disease include generalized lymphadenopathy, erosions of mucous membranes, hemorrhages, enlarged liver, and ascites. The internal lymph nodes show less enlargement than the superficial lymph nodes already mentioned. Splenomegaly is usually seen in acute cases, with the spleen enlarged up to four times normal size.

In addition to the mucosal erosions seen clinically, irregular linear erosions have been decribed in the esophagus. Edema, petechiae, erosions and ulcers in the abomasum, and petechial and ecchymotic hemorrhages in the small and large intestines have also been described. One author described linear hemorrhages along the longitudinal folds of the duodenal musosa in seven cases.

The liver is usually described as slightly enlarged or swollen and yellowish in color, with a "nutmeg" appearance. The gall bladder is usually enlarged and "paint-brush" hemorrhages have been described on the serosal surface.

Histopathologically, a lymphoproliferative disorder with mononuclear infiltrations is the usual picture. Proliferative endotheliosis is considered pathognomonic.

Petechial or ecchymotic "paint-brush" type hemorrhages have often been described on the epicardium and endocardium.

Kidneys are often pale or yellowish brown in color, with petechial hemorrhages. Blood clots in the renal pelvis and infarcts have been noted. The urinary bladder may have petechial and ecchymotic hemorrhages, or both.

Ascitic fluid in the abdominal cavity, up to 5 liters, has been reported.

Diagnosis

Because the etiological agent of Jembrana disease is unknown, diagnosis is based on history, clinical signs, gross pathology, and histopathology. During the more than 20 years different investigators have been studying this disease, many tests have been performed in attempts to identify a known disease antigen. These include examinations of paired sera for antibodies to rinderpest (measles hemagglutination-inhibition test) and human rickettsia (Weil-Felix test). Suspensions of lymph node and spleen from natural cases were tested against serums for rinderpest virus (agar gel diffusion test), Coxiella burnetii, Ehrlichia phagocytophilia, and Cowdria ruminantium. No antigenic relationship was detected between Jembrana disease and these reagents. Animal diseases that have been considered in the differential diagnosis of Jembrana disease include rinderpest, bovine viral diarrhea, hemorrhagic septicemia, malignant catarrhal fever, theileriosis (East Coast fever, Theileria annulata infection, etc.), bovine petechial fever (Ehrlichia ondiri infection), bluetongue, Ibaraki, and foot-and-mouth disease. All investigators, including veterinarians of the Government of Indonesia, seem convinced that Jembrana disease is not identical with any other known disease entity. However, based on laboratory work a rickettsia (Ehrlichia sp.) seems to be emerging as the most probable causative organism, according to Dr. A. A. Ressang, Team Leader of the Bali Cattle Disease Investigation Unit (BCDIU), in Denpasar, Bali.

Transmission

Jembrana disease can be transmitted to Bali cattle using infective blood, plasma, and tissue suspensions from natural cases. The experimental disease was found to be similar in all respects to the natural disease. The incubation period varied from 2 to 10 days. The disease was experimentally transmitted to buffaloes, sheep, and goats but not to pigs.

Experimental evidence that Boophilus microplus ticks have transmitted the disease is equivocal and is not supported by field studies. A total of 32 arthropods species have been associated with Bali cattle in the field. Of these, Culicoides (biting midges) were considered to be worthy of additional study as possible Jembrana disease vectors. Ranked according to apparent population density, they were C. peregrinus, C. schultzei, C. amamiensis, C. matsuqawai, C. subflavescens, and C. ebeli. Some C. humeralis were trapped. Fourteen species of mosquitoes were also trapped. Preliminary studies have failed to incriminate any of these species as a vector of Jembrana disease. However, many investigators suggest that it is most probably a vector-borne disease.

Treatment

Veterinarians who first began working with Jembrana disease felt that tetracycline effectively reduced the course of the disease. Many trials have been conducted with various antibiotic combinations such as Oxyvet, Leucomycin (chloramphenicol), Ertiten (chloramphenicol, corticosteroid, and antihistamine), and Tetrachlorina (chloramphenicol, oxytetracycline, and ascorbic acid). Results indicate that antibiotics do not alter the course of the disease, although secondary bacterial infections may be reduced.

Epidemiological Factors

No significant correlation was found in attempts to relate morbidity and mortality rates to season, altitude, rice paddies, cattle population density, and buffalo population density.

Impact on Animal Production

Jembrana disease has been the cause of major economic loss on Bali Island and is of grave concern to both Balinese and other Indonesian livestock producers. The presence of the disease has curtailed exports of cattle and buffalo from Bali, except males for slaughter, and has caused the direct loss to the farmer of animals vital to the production of rice and generation of personal income. (Dr. James T. Cavanaugh, Veterinary Attache, U.S. Embassy, Manila, The Philippines.)

Asiatic Hemorrhagic Septicemia Addendum

Asiatic hemorrhagic septicemia, a disease considered to be foreign to the United States, was reviewed in the June 1985 issue (13-2). The article mentioned only one occurrence of Pasteurella multocida serotype 6:B in bison in the United States. That occurrence was first reported in Texas by K. L. Heddleston, K. R. Rhoades, and P. A. Rebers in 1967 (Am. J. Vet. Res. 28,125:1003-1012). Serotype 6:B was also isolated from two calves in Pennsylvania in 1968 (D. C. Kradel, K. L. Heddleston, J. V. Risser, and J. E. Manspeaker. Vet. Med./Small An. Clin., Feb. 1969:145-147). (Editor).

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